Methyl Mercaptan and Methyl Mercaptide – Comments of Environmental Defense

(Submitted via Internet 4/19/02)

Environmental Defense appreciates this opportunity to submit comments on the robust summary/test plan for methyl mercaptan and methyl mercaptide.

The test plan submitted by the Mercaptans/Thiols Council (MTC) is well written and easy to follow. Methyl mercaptan (MeSH) is used as an intermediate in producing jet fuel and in the synthesis of a number of agents including some pesticides. The sponsor proposes that MESH and methyl mercaptide (NaMeSH) be considered as analogs and that the test data between the two agents are interchangeable. This proposal is convincing and we agree with it along with the statement that NAMeSH be used in further testing because it is safer and easier to handle.
In regards to environmental fate and ecotoxicity studies, we agree with MTC's proposal to generate photodegadation estimates, an acute fish toxicity study, a daphnia study, and an algal inhibition study. We also agree that existing data on acute toxicity, repeat dosing, and genetic toxicity are adequate to fulfill HPV requirements. MTC proposes that data from a reproductive/developmental study on hydrogen sulfide (H2S) can be used to fill this requirement for MeSH. They contend that H2S and MeSH have similar physiochemical characteristics, metabolism and mechanisms of toxicity. We have several concerns about this proposal as summarized below:

1. While we agree that the mechanism of acute toxicity (lethality) is likely a consequence of cytochrome oxidase inhibition for both H2S and MeSH, this does not mean that the mechanism is the same for non-lethal endpoints such as central nervous system effects. There is no data that shows a common mechanism for non-lethal effects. For example, H2S can directly stimulate chemoreceptors of the cardiovascular system to cause cardiac irregularities.

2.The developmental/reproductive studies conducted at CIIT on H2S are of high quality and the authors conclude that H2S is neither a developmental nor a reproductive toxicant at doses as high as 80 ppm. However, the CIIT studies (Brennerman 2000) also showed that H2S caused olfactory neuron loss and nasal cell hyperplasia at doses of 30 and 80 ppm. The mechanism of action responsible for this effect is not known.

3. There are numerous papers in the scientific literature on H2S toxicology. Most of these are summarized by EPA in the January 2000 public draft on H2S. These studies reveal that H2S may cause a wide variety of effects including changes in eye cytology, EEG activity and several brain enzymes. Developmental/reproductive studies not reported by MTC in the test plan include effects on serotonin levels and Purkinje cells.

4. If the sponsor is allowed to use the CIIT H2S studies to fill the reproductive/developmental endpoint for MeSH, then it should be assumed that all effects caused by H2S would also be caused by MeSH. One option for MTC would be to conduct a reproductive/developmental study on MeSH.

5. The sponsor states on page 18 that the ACGIH TWA is 10 ppm. We understand that ACGIH is in the process of lowering that number to 5 ppm or less. This value is much higher than other AAL's or regulations on acceptable ambient levels. For example, California's ambient air standard is 0.03 ppm and North Carolina is proposing to lower their AAL from 1.5 ppm to 0.04 ppm based on recent studies showing that H2S induces asthmatic responses and causes ocular toxicity and irritation.

Thank you for this opportunity to comment.

George Lucier, Ph.D.
Consulting Toxicologist, Environmental Defense

Karen Florini
Senior Attorney, Environmental Defense